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Intraoperative problems after cardiopulmonary bypass

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Literature review current through: **Apr 2026.**
This topic last updated: **Jan 15, 2025.**

INTRODUCTION

Cardiovascular and other systemic perturbations are frequently encountered immediately after weaning from cardiopulmonary bypass (CPB). These are often predictable based upon patient-specific and cardiac surgical procedure-specific factors. However, some patients experience unpredictable, sudden, or severe complications that require immediate intervention and/or an urgent reinstitution of CPB. Patients successfully weaned from CPB may continue to have challenges during the early postbypass period.

This topic will discuss intraoperative problems that are commonly encountered in the immediate postbypass period. A detailed discussion of management of bleeding and coagulopathy after CPB is available in a separate topic. (See "Achieving hemostasis after cardiac surgery with cardiopulmonary bypass".)

Patient management during CPB and the process of weaning from CPB support are described in separate topics. (See "Management of cardiopulmonary bypass" and "Weaning from cardiopulmonary bypass".)

Problems in the immediate postoperative period are discussed separately. (See "Postoperative care after cardiac surgery".)

CARDIOVASCULAR PROBLEMS

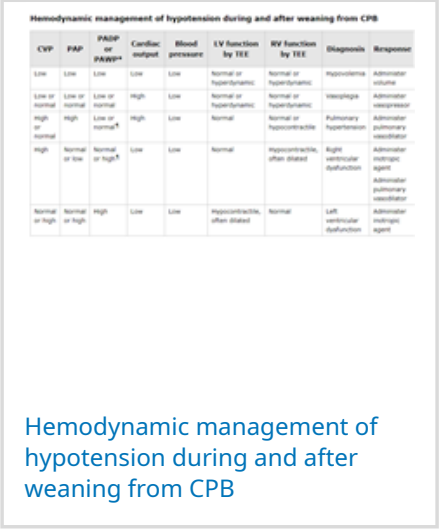
General considerations — Cardiovascular instability can occur immediately after cardiac surgical procedures requiring CPB, often with resultant hypotension [1]. In this setting, clinically significant hypotension is usually due to one or more of the following problems:

- Inadequate **preload**, which impairs filling of the left ventricle (LV)
- Compromised **contractility**, which may be due to either global or focal LV or right ventricle (RV) dysfunction
- Decreased **afterload**, which manifests as reduced systemic vascular resistance (SVR) and vasoplegia
- Too low or excessively high **heart rate** (HR)

- **Rhythm** other than sinus rhythm, with loss of atrioventricular (AV) synchrony

Before initiating treatment of hypotension, it is important to determine whether a central to peripheral arterial pressure gradient accounts for the appearance of low blood pressure (BP) on the monitored arterial tracing (figure 1). This can be accomplished by inserting a new femoral arterial monitoring cannula, or by direct transduction of the central aortic root pressure through the aortic or cardioplegia cannulae already in situ. These non-physiologic gradients commonly occur following CPB, and consequently, therapy targeted to treat a low radial artery pressure may be inappropriate. In one prospective study, the arterial pressure in the radial artery underestimated the femoral pressure in 34 percent of 435 patients, with a systolic BP difference ≥25 mmHg or a mean arterial pressure (MAP) difference ≥10 mmHg [2]. Such significant gradients were more likely in patients with smaller body surface area, longer aortic cross-clamping time, less fluid administration, or a preoperative history of hypertension.

A general guide to treatment of hypotension during and after weaning from CPB is described in the table (table 1), and greater detail is provided below.



Hemodynamic management of hypotension during and after weaning from CPB

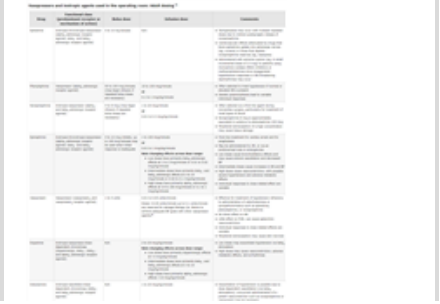
Table 1 - larger image below

Left ventricular dysfunction — LV dysfunction may occur due to preexisting chronic ventricular dysfunction, and may be exacerbated by myocardial ischemia, stunning, or ischemia/reperfusion injury occurring during CPB with cardioplegia-induced cardiac arrest. (See "Postoperative complications among patients undergoing cardiac surgery", section on 'Cardiac dysfunction'.)

Myocardial injury resulting in postbypass ischemia or infarction is particularly likely in operations involving revascularization for coronary artery disease. Other causes of myocardial dysfunction include incomplete myocardial preservation during CPB, particulate or air emboli in a coronary graft or a native coronary artery, coronary spasm, coronary thrombosis, technical surgical problems such as kinking of a graft or compromised graft anastomosis, or incomplete revascularization secondary to distal disease or inoperable vessels. Transesophageal echocardiography (TEE) is employed to assess global LV function as well as regional wall motion abnormalities that may be associated with specific graft or vessel compromise. (See "Postoperative complications among patients undergoing cardiac surgery", section on 'Mechanical complications'.)

After potentially reversible surgical factors (eg, graft kinking) are investigated and addressed, interventions by the anesthesiologist should focus on optimizing HR, pacing mode, as well as vasoactive and/or inotropic drug therapy.

Vasoactive drug therapy — We do not routinely administer inotropic and vasopressor drugs without an indication such as preexisting myocardial dysfunction or significant vasoplegia during CPB. Usually, LV dysfunction will improve with inotropic drug therapy or combination therapy with positive inotropic and vasodilator agents to optimize cardiac index (CI) [3-6]. However, there are marked institutional variations in the selection of specific



vasoactive agents and combinations of agents [7-12]. Initially, small boluses of ephedrine (5 to 10 mg) or diluted epinephrine (5 to 10 mcg) may be administered to treat ventricular dysfunction and hypotension; if necessary, infusion of one or more vasoactive agents is initiated (table 2). (See "Weaning from cardiopulmonary bypass", section on 'Maintenance of optimal pacemaker function' and "Postoperative complications among patients undergoing cardiac surgery", section on 'Inadequate inotropy'.)

Vasopressors and inotropic agents used in the operating room: Adult dosing*¶

Table 2 - larger image below

For inotropic support, infusions of sympathomimetic amines (eg, epinephrine, dobutamine, dopamine) are commonly used as first-line therapies, alone or in combination with milrinone [3,10,11]. The inodilator milrinone is a phosphodiesterase inhibitor that produces positive inotropic effects by slowing hydrolysis of cyclic adenosine monophosphate and works independently of beta-adrenergic receptors [5,6,13-16]. Since administration of milrinone may significantly reduce SVR, concomitant use of an arterial vasoconstrictor (eg, phenylephrine, vasopressin, norepinephrine) may be necessary. Selection of a first-line inotropic agent depends on the individual patient's hemodynamic abnormalities during the weaning process (table 1 and table 2), as well as institutional preferences [3,4,7,9-11,17]. Alternate inotropic agent(s) may be selected as conditions change during the postbypass period. (See "Use of vasopressors and inotropes".)

The majority of cardiac surgical patients do receive at least one inotropic agent during their case (52.9 percent) [18]. Agent selection is primarily related to the individual patient's condition (70.6 percent) but factors such as the preference of the anesthesiologist (6.8 percent) and institutional practice (22.6 percent) may impact use and agent selection. Administration of inotropic and other vasoactive agents after cardiac surgery is generally based on achieving short-term hemodynamic goals, particularly in patients with low preoperative LV ejection fraction (EF; eg, <30 percent) [3,7]. However, benefits versus harms of prophylactic administration have not been established [18-20]. A 2024 systematic review of randomized trials (29 studies with 3307 participants) examined prophylactic use of inotropic agents to prevent low cardiac output syndrome and mortality during cardiac surgery [19]. Due to study limitations that included small size, imprecision, and inconsistency, data were inadequate to suggest any benefit (or harm) for most inotropic agents (milrinone, amrinone, dobutamine, and dopamine) compared with placebo or any other agent. However, low certainty evidence suggested that prophylactic infusion of levosimendan may reduce the incidence of low cardiac output syndrome (risk ratio [RR] 0.43, 95% CI 0.25-0.74; six studies with 1724 participants) and mortality (RR 0.65, 95% CI 0.43-0.97; 14 studies with 2348 participants) compared with placebo [19].

Levosimendan is an inodilator that increases myocardial sensitivity to calcium, which increases cardiac contractility and opens adenosine triphosphate channels in vascular smooth muscle causing vasodilation [21]. Although not available in the United States, levosimendan is often used during weaning from CPB and/or in the postoperative period in countries where it is available [22]. While meta-analyses that included mainly small trials, these have suggested a benefit of levosimendan on survival and adverse events in patients with EF (ie, <40 percent) after cardiac surgery, data have been limited by study size, heterogeneity, and risk of bias [19,22,23]. The largest randomized multicenter trial was conducted in 849 cardiac surgical patients; prophylactic levosimendan administration (with infusion initiated shortly before surgery and

continued for 23 hours) did not result in a lower rate of death, perioperative myocardial infarction, kidney replacement therapy, or use of a ventricular assist device (VAD) compared with placebo [24].

Conversely, some data suggest that perioperative use of inotropes is associated with increased postoperative mortality and morbidity in cardiac surgical patients [7,25-27]. As an example, in one observational study using propensity score matching, perioperative use of inotropes was independently associated with more than threefold increased mortality (adjusted hazard ratio [HR] 3.7, 95% CI 2.1-6.5) [7]. However, the investigators acknowledged that use of inotropic therapy may be the only option for weaning from CPB in some cases. Inferring causality in this association is misleading.

In patients who are hypertensive during weaning from bypass, nitroglycerin is typically administered if concomitant ischemia is suspected, while other direct-acting vasodilators such as clevidipine, nicardipine, milrinone, or nitroprusside may be helpful to reduce afterload in patients without ischemia.

Left ventricular diastolic dysfunction — Diastolic dysfunction results in decreased LV filling due to impaired myocardial relaxation. This may be caused by prior myocardial stunning from earlier cardioplegia delivery or other preexisting LV conditions such as hypertrophy, fibrosis, or infiltrative disease. Diastolic dysfunction often occurs in conjunction with systolic dysfunction but can be present in some patients who have preserved LV systolic function and EF. Furthermore, new or ongoing intraoperative ischemia and/or hypoxia may also result in functional abnormalities of myocyte relaxation and new diastolic dysfunction. (See "Pathophysiology of heart failure with preserved ejection fraction".)

Weaning from CPB may be challenging in patients with diastolic dysfunction. The following considerations are applicable [28]:

- Euvolemia should be maintained. Appropriate interpretation of ventricular filling pressures (eg, central venous pressure (CVP) or pulmonary artery wedge pressure [PAWP]) is important since patients with diastolic dysfunction may have abnormally increased filling pressures for any given LV or RV filling volume. As a result, transient and abrupt elevation in filling pressures should not be overtreated with excessive diuresis or preload reduction that may result in precipitous hypotension. However, excessive volume administration may precipitate pulmonary edema due to impaired LV compliance. (See "Postoperative complications among patients undergoing cardiac surgery", section on 'Inadequate preload'.)

- Optimal HR should be maintained with atrial or AV sequential pacing at 80 to 90 beats per minute (bpm). Occasionally, a slightly faster HR of 90 to 100 bpm is selected (eg, for patients with frequent premature atrial contractions [PACs] to best maintain a stable rhythm).

- AV synchrony should be maintained, since the atrial contribution to ventricular filling may exceed 30 percent, particularly in patients with early stage (Grade 1) diastolic dysfunction (see "Echocardiographic evaluation of left ventricular diastolic function in adults", section on 'Diagnosis of diastolic dysfunction'). Atrial pacing or AV sequential pacing may be needed to achieve this goal.

Right ventricular dysfunction — RV dysfunction may complicate weaning from CPB [1]. Dysfunction may be preexisting, or caused or exacerbated by pulmonary hypertension, RV ischemia or infarct, intracoronary or pulmonary air embolism, or tricuspid regurgitation [29]. Intracardiac air, always present to some degree following open left-sided cardiac procedures, preferentially enters the right coronary artery (RCA) due to its

more anterior (non-dependent) position in the supine patient and often results in significant RV ischemia and dysfunction. (See 'Arterial air embolization' below.)

RV dysfunction manifests as increased CVP with systemic hypotension due to reduced LV preload. Severe RV dysfunction may also compromise LV perfusion. On direct visual inspection of the surgical field, the RV appears distended and shows poor contractility. TEE examination reveals severe RV hypokinesis often accompanied by significant tricuspid regurgitation. Also, the interventricular septum may be shifted towards the left, thereby impairing filling of the LV and resulting in increased left-sided filling pressures (ie, PCWP) despite inadequate LV filling volume. Leftward septal shift during diastole indicates RV volume overload, while leftward septal shift during systole indicates RV pressure overload. Some clinicians use RV pressure waveform analysis to facilitate diagnosis of systolic and diastolic RV dysfunction [30]. (See "Echocardiographic assessment of the right heart".)

Management includes ensuring that systemic BP and coronary perfusion pressure are adequate, as well as preventing increased pulmonary vascular resistance (PVR), which can be caused by hypoxemia, hypercarbia, acidosis, hypothermia, or pain. Excessive administration of fluid is avoided, as this may worsen RV dysfunction. Pharmacologic therapies for RV dysfunction provide inotropic support with intravenous agents that produce pulmonary arterial vasodilation (eg, milrinone, dobutamine) [31]. Levosimendan has also been used to support the RV in this setting [32]. Typically, such agents are combined with administration of vasopressor therapy (eg, norepinephrine, vasopressin) to maintain systemic perfusion pressure.

In refractory RV failure, continuous inhalation of an aerosolized vasodilator to treat pulmonary hypertension and reduce RV afterload is reasonable [1]. Typical agents and doses are:

- **Nitric oxide** – Administration of 5 to 20 parts per million (ppm) requires a nitric oxide administration apparatus, a nitric oxide gas analyzer, and a nitrogen dioxide gas analyzer, as well as a specially trained health care professional. Doses >20 ppm should be used with caution; doses >40 ppm provide minimal additional clinical benefit. Adverse effects of high doses administered for prolonged periods include development of methemoglobinemia, and abrupt discontinuation may cause rebound pulmonary hypertension. (See "Inhaled nitric oxide in adults: Biology and indications for use".)

- **Epoprostenol** – Administration of epoprostenol is accomplished via a nebulizer in the ventilator circuit; typical doses in the postbypass setting are 30 to 50 ng/kg/minute [33].

- **Milrinone** – Inhaled milrinone has been used for acute RV failure in several studies; although apparently safe, its efficacy in treating acute RV failure or pulmonary hypertension via this route of administration is uncertain [16,34].

- **Combined inhaled milrinone and epoprostenol** – In one study of 128 consecutive patients with pulmonary hypertension or RV dysfunction after CPB, inhaled milrinone and epoprostenol administered in combination before CPB resulted in a positive pulmonary vasodilator response in 77 percent of patients [35]. Also, use of inotropic agents in the postbypass period was lower in patients who responded to treatment (8.1 versus 27.6 percent).

Meta-analyses conducted in 2017, 2018, and 2019 have not shown benefits in clinical outcomes when inhaled aerosolized pulmonary vasodilators were employed during cardiac surgery compared with intravenously administered agents [16,36,37].

In rare cases of severe RV dysfunction, a patient may require a temporary mechanical RV assist device.

Vasoplegia — Severe systemic vasodilation (ie, vasoplegia) with markedly decreased SVR may become evident during or after CPB in 5 to 25 percent of patients undergoing cardiac surgery, and may cause severe persistent postbypass and postoperative hypotension despite a normal or increased CI [38-42]. Risk factors for vasoplegia include preoperative use of certain medications (eg, angiotensin-converting enzyme [ACE] inhibitors, calcium channel blockers, diuretics), patient-related factors such as dialysis-dependent kidney failure or recent myocardial infarction, and procedure-related factors such as prolonged bypass times for complex operations, prebypass hemodynamic instability, selected cardiac valve procedures (mitral, pulmonary, or tricuspid valves), or insertion of a left VAD [38,39,41,43,44].

Agents used for treatment include norepinephrine and vasopressin. Most patients with vasodilatory shock (ie, distributive shock) respond to hemodynamic-guided IV fluid therapy and/or IV norepinephrine, often at low doses [45,46]. However, adding vasopressin may be necessary for effective treatment when vasoplegia is refractory to catecholamines [41,47]. In a 2018 meta-analysis of eight trials that included 625 patients undergoing cardiac surgery, use of vasopressin reduced risk of vasodilatory shock (odds ratio [OR] 0.4, 95% CI 0.16-0.97), atrial fibrillation (OR 0.42, 95% CI 0.21-0.82), and overall perioperative complications (OR 0.33, 95% CI 0.20-0.54), compared with standard therapy or placebo [48]. Another 2018 meta-analysis in critically ill patients with vasodilatory shock also noted that the addition of vasopressin to catecholamine therapy was associated with lower rates of atrial fibrillation compared with administration of catecholamines alone (risk ratio [RR] 0.77, 95% CI, 0.67-0.88) [49].

Other agents that have been used to treat refractory vasoplegia include methylene blue, angiotensin II, vitamin C, or hydroxycobalamin (table 3) [40,47,50-66]. Further discussion of management of vasoplegia during and after CPB is available in other topics. (See "Management of cardiopulmonary bypass", section on 'Management of hypotension' and "Postoperative complications among patients undergoing cardiac surgery", section on 'Vasodilatory shock'.)

Vasopressors used to treat vasoplegic syndrome

Table 3 - larger image below

Arrhythmias — Both supraventricular and ventricular arrhythmias are common during and after weaning from CPB. Normal sinus rhythm is ideal because it provides an atrial contribution to ventricular filling and normal, synchronized contraction of the ventricles.

- **Ventricular arrhythmias** – Ventricular fibrillation during weaning or the postbypass period should be immediately treated with defibrillation. In the open chest, internal paddles are applied directly to the heart to deliver 10 to 20 joules of electricity. If ventricular arrhythmias persist or recur, an antiarrhythmic drug infusion, usually amiodarone, is initiated. (See "Cardioversion and defibrillation for specific arrhythmias" and "Amiodarone: Clinical uses, administration, and adverse effects", section on 'Ventricular arrhythmias'.)

  Factors such as hypokalemia and hypomagnesemia that may contribute to development of arrhythmia should be corrected. Persistent or recurrent ventricular fibrillation may indicate myocardial ischemia and inadequacy of coronary blood flow.

- **Atrial fibrillation** – Atrial fibrillation (AF) is the most common arrhythmia after cardiac surgery but usually develops two to five days postoperatively rather than in the immediate postbypass period. AF or atrial flutter that occurs in the immediate postbypass period can often be converted to normal sinus rhythm with synchronized internal cardioversion prior to chest closure., especially if sinus rhythm was present before CPB [67]. (See "Cardioversion and defibrillation for specific arrhythmias" and "Atrial fibrillation: Cardioversion".)

  If cardioversion is unsuccessful, a reasonable option is amiodarone 150 mg bolus IV administered over 10 minutes and followed by continuous infusion at 1 mg/minute. If rate control is necessary, esmolol, metoprolol, or diltiazem may be administered [68]. (See "Atrial fibrillation in adults: Rate control in patients without heart failure" and "Atrial fibrillation: Cardioversion" and "Amiodarone: Clinical uses, administration, and adverse effects", section on 'Atrial fibrillation'.)

- **Bradycardia** – If sinus bradycardia, complete heart block, or asystole develops, temporary pacing via surgically placed epicardial wires should be instituted. Sinus bradycardia is preferentially treated by atrial pacing. However, if heart block is present, AV sequential pacing is employed to maintain atrioventricular synchrony, which promotes adequate filling of the LV.

  If there is no organized atrial rhythm (eg, AF) and HR is slow, then ventricular pacing must be used. In such cases, absence of AV synchrony limits CI, particularly in patients with diastolic dysfunction and reduced LV compliance.

  When bradycardia or heart block is dependent on external pacing, the capture threshold of the pacemaker should be assessed. High thresholds should prompt wire replacement, repositioning, or placement of a second set of wires in the case of pacemaker lead failure. (See "Postoperative complications among patients undergoing cardiac surgery", section on 'Dysrhythmias'.)

  Biventricular pacing has been demonstrated to improve cardiac output during weaning from CPB in patients with heart failure, compared with standard pacing, but is more complex and not commonly employed in this setting [69].

Arterial air embolization — Air may accumulate in the left heart chambers during procedures where the left-sided chambers of the heart are opened (eg, aortic or mitral valve repair or replacement, ascending aorta repair, ventricular septal defect repair). Even procedures not typically associated with opening the left heart chambers may be associated with entrainment of air, including injury associated with ligation of the left atrial appendage. Intracardiac air may embolize to the coronary, cerebral, and peripheral circulations once the aortic cross-clamp is removed and the heart begins to contract (movie 1A-D). Since the RCA is in an anterior or nondependent position when a patient is supine, air preferentially enters the RCA causing ischemia of the RV and inferior LV wall (right dominant circulation) (movie 1A), as well as arrhythmias, particularly heart block. Air microembolization to the cerebral circulation has been associated in some studies with neurologic dysfunction as well as seizures [70].

TEE is employed to identify air in the heart during the weaning process and guide its removal primarily by surgical venting of the LV via a vent placed through the right upper pulmonary vein or aortic root via the antegrade cardioplegia cannula, thereby reducing arterial embolic load. Inotropic or vasopressor drug treatment may be necessary to increase BP and improve coronary perfusion until residual air has been cleared from the heart and coronary vessels.

Surgical or technical problems — Surgical or technical problems resulting in myocardial ischemia are a rare cause of difficulty in weaning. Examples include poor quality of a coronary bypass graft anastomosis or kinking of the graft, embolization of air or microparticulate debris into a native coronary artery or graft, or suture ligation or injury of a coronary artery during aortic valve, aortic root, or mitral valve replacement (image 1). In such cases, myocardial ischemia is identified by ventricular wall motion abnormalities noted on TEE examination, as well as hypotension and low cardiac output (CO) despite high doses of inotropic and/or vasopressor agents, and/or ST-segment changes on the electrocardiogram (ECG). Suspected poor coronary graft flow after coronary revascularization procedures may be confirmed with a Doppler flow probe applied to the graft. (See "Postoperative complications among patients undergoing cardiac surgery", section on 'Mechanical complications'.)

Iatrogenic aortic dissection is a rare complication of cardiac surgery; a retrospective report described seven cases among 3000 cardiac surgical procedures in a single institution [71]. Aortic dissection usually occurred due to aortic cannulation and was detected at the onset of CPB (see "Initiation of cardiopulmonary bypass", section on 'Aortic cannulation'), but is sometimes diagnosed with TEE at the end of bypass following decannulation (image 2 and image 3) (see "Anesthesia for coronary artery bypass grafting surgery", section on 'Post-revascularization transesophageal echocardiography'). Further TEE assessment should include the anatomic location and extent of the dissection and whether there is concomitant involvement of the aortic valve. Additional discussion of the surgical and anesthetic management of this condition is available separately. (See "Anesthesia for aortic surgery with hypothermia and elective circulatory arrest in adult patients", section on 'Considerations for emergency surgery for acute aortic dissection' and "Surgical and endovascular management of acute type A aortic dissection", section on 'Surgical repair'.)

Left ventricular outflow tract obstruction — LV outflow tract (LVOT) obstruction can occur when the outflow tract is narrowed by LV septal hypertrophy and/or abnormal length and positioning of the anterior mitral valve leaflet. The resultant systolic anterior mitral leaflet-septal contact causes mechanical obstruction to LV ejection, as well as mitral regurgitation due to inadequate coaptation of the mitral valve leaflets (image 4 and movie 2) [72]. Such LVOT obstruction may be preexisting or may occur after mitral valve repair, aortic valve replacement, and other cardiac operations in patients with severe underlying left ventricular hypertrophy. LVOT obstruction may also occur in otherwise low risk patients during periods of significant hypovolemia.

LVOT obstruction is dynamic. Because a hypertrophied LV has reduced compliance and is extremely sensitive to changes in preload and afterload, hypovolemia or reduced SVR will worsen obstruction. Since tachycardia or a hypercontractile state will also worsen obstruction, administration of sympathomimetic drugs to treat hypotension is relatively contraindicated.

Treatment of hemodynamically significant LVOT obstruction includes:

- Increasing LV volume with fluid administration.
- Increasing SVR with vasoconstrictors that do not have inotropic or chronotropic properties (eg, phenylephrine or vasopressin).
- Decreasing inotropy and HR with anesthetic agents or beta-blockers. Epicardial pacing can maintain an adequate HR when these therapies cause bradycardia.

TEE interpretation is critical for identification of LVOT obstruction and optimal intraoperative management because standard cardiovascular monitoring will not reveal the underlying cause of hypotension and often leads to inappropriate therapy. For example, systemic hypotension with low CO and mitral regurgitation is often treated with inotropic agents, but such treatment only worsens LVOT obstruction and further decreases BP. Also, TEE assessment of the mechanism causing LVOT obstruction may lead to a surgical decision to reinstitute CPB to revise a mitral valve repair or perform ventricular septal myectomy.

Cardiogenic shock — Shock that is refractory to medical intervention after cardiac surgery is referred to as postcardiotomy cardiogenic shock [73]. This occurs in 0.2 to 6.0 percent of patients after cardiac surgery with CPB, and manifests as the inability to maintain adequate CO, oxygenation, or end-organ perfusion [74-76]. Temporary mechanical circulatory support (eg, intraaortic balloon pump [IABP] counterpulsation, percutaneous or implantable VAD, extracorporeal membrane oxygenation [ECMO]) may be employed in cases of refractory ventricular dysfunction resulting in persistently low CO [74]. Selection of a circulatory assist device depends on individual patient hemodynamic factors, surgical preferences, and institutional resources (table 4). Contraindications and complications associated with all temporary mechanical support devices are listed in the table (table 5).

Short-term mechanical circulatory assist devices

- **Intraaortic balloon pump** – IABP counterpulsation is a mainstay for treatment of failure to wean from CPB, with between 14 and 28 percent of all IABP use occurring after CPB [77-83]. Several studies have been performed assessing the effectiveness of an IABP during weaning from CPB, with in-hospital mortality ranging from 20 to 34 percent [77,80-83]. In various studies, risk factors for mortality have included high vasopressor doses, mixed venous saturation <60 percent, higher left atrial pressure or LVEDP, higher preoperative creatinine, longer aortic cross-clamp duration, and urgent/emergency surgery. Other complications include limb ischemia, vascular injury, and, rarely, significant hemorrhage. Risk factors for complications include peripheral artery disease, older age, female sex, small body surface area <1.8 m^2, larger catheter size (>9.5 French), diabetes mellitus, hypertension, prolonged need for IABP support, and CI <2.2 L/minute/m^2. (See "Intraaortic balloon pump counterpulsation".)

- **Ventricular assist devices** – A temporary VAD can provide circulatory support and allow time for myocardial recovery [84]. In rare instances, the VAD is inserted to serve as a bridge to urgent cardiac transplantation [84-86]. Efficacy of VADs for management of cardiogenic shock developing during cardiac surgery has been demonstrated in case reports and small clinical trials [86-88]. (See "Short-term mechanical circulatory assist devices".)

Ideally, TEE or fluoroscopy guidance is used to confirm the indication for support placement, evaluate for contraindications to a particular device, and confirm proper placement of inflow and outflow cannulae and the device itself, as well as to assess device function after placement, as discussed in detail in a separate topic. Findings such as aortic dissection may necessitate continuation of medical management while a safe cannulation strategy for mechanical support is implemented (image 3). (See "Short-term left ventricular mechanical circulatory support: Use of echocardiography during initiation and management".)

Extracorporeal membrane oxygenation — In some patients with concomitant respiratory failure and LV failure that cannot be managed with changes in ventilation strategies and medications, ECMO may be selected to provide both hemodynamic and respiratory support. Components of an ECMO circuit include the

gas exchange unit (oxygenator/heater), driving force (pump), and tubing. Similar to CPB, ECMO can support oxygenation, ventilation, circulation, heating, and cooling, but unlike CPB, ECMO lacks a venous reservoir and the ability to administer fluids directly via the circuit. Typically, ECMO is easier to establish than mechanical ventricular support with an Impella VAD and allows higher flow rate (up to 10 L/minute). (See "Short-term mechanical circulatory assist devices", section on 'Extracorporeal membrane oxygenation' and "Extracorporeal life support in adults in the intensive care unit: Overview".)

Cannulation for ECMO may be either peripheral or central (image 5), typically with TEE guidance, as described in detail in a separate topic (see "Extracorporeal life support in adults in the intensive care unit: The role of transesophageal echocardiography (TEE)"). Advantages to peripheral cannulation include ease of chest closure and removal of cannulae, as well as the fact that peripheral cannulae are generally more secure (less likely to dislodge) than central cannulae. However, central cannulation for VV or VA ECMO is often easier, allows larger cannula, and eliminates the risk of femoral venous or arterial complications, including peripheral ischemia associated with a large cannula in the femoral artery. The major disadvantage is that central cannulae are less secure and dislodgement may cause life-threatening hemorrhage.

Initial settings for ECMO include a target blood flow rate of 60 mL/kg/minute in patients with minimal cardiac function [89]. Mean arterial pressure should be maintained between 65 and 80 mmHg [89]. Once the patient is stable without bleeding, a postoperative heparin infusion may be administered to keep the partial thromboplastin time at approximately 1.5 to 2 times normal [90].

Two 2018 meta-analyses of observational studies in patients who received ECMO for postcardiotomy cardiogenic shock (one that included 31 studies and 2986 patients and the other with 20 studies and 2877 patients) have noted that approximately one-third survived to hospital discharge [91,92]. Factors associated with survival in these meta-analyses included younger age and lower baseline blood lactate levels. Prediction of outcome after ECMO insertion in this setting is challenging due to heterogeneity of specific patient- and procedure-related reasons for failure to wean from CPB.

Early postbypass cardiac arrest — Cardiac arrest may occur at any time in the postbypass period, usually in the first five postoperative hours [93,94]. In one series of 3982 patients undergoing cardiac surgery, the incidence was 0.7 percent in the first 24 hours after surgery [95].

Arrest immediately after CPB may be the result of ongoing myocardial ischemia due to inadequate revascularization, delayed entry of air into the coronary circulation resulting in ischemia, loss of pacing capability in a pacing-dependent patient, low CI, mechanical impediments to cardiac function (eg, tamponade or graft inadequacy), or major bleeding [93-95].

PULMONARY PROBLEMS

We use a lung protective ventilation strategy in the postbypass period (with low tidal volume [TV] <8 mL/kg ideal body weight, positive end-expiratory pressure [PEEP] ≥5 cmH_2O, and low driving pressure [peak inspiratory pressure PEEP] <16 cmH_2O) to potentially reduce the incidence of postoperative pulmonary complications. These ventilator settings are consistent with recommendations for lung-protective ventilation for all patients undergoing anesthesia and surgery with the use of mechanical ventilation. (See "Mechanical ventilation during anesthesia in adults", section on 'Lung protective ventilation during anesthesia'.)

Airway obstruction — Airway occlusion, bronchospasm, and respiratory insufficiency may result in hypoxemia, inadequate ventilation, and increased airway pressures, as well as increased pulmonary vascular resistance with resultant right ventricular (RV) dysfunction.

The anesthesiologist should be aware of any difficulties in ventilating the lungs before attempting to wean from CPB. Both lungs should be expanded with positive pressure ventilation, and appropriate lung inflation should be confirmed by direct observation in the open chest. The differential diagnosis of poor lung inflation and/or deflation includes airway obstruction (eg, kinked endotracheal tube [ETT], tracheal or bronchial obstruction [eg, mucous plug], airway compression by the transesophageal echocardiography [TEE] probe), right mainstem intubation, or pulmonary aspiration. Blood in the airway is uncommon, but may occur due to pulmonary parenchymal injury, pulmonary artery rupture caused by a pulmonary artery catheter (PAC), or injury during pulmonary thromboendarterectomy.

An initial step for evaluation of airway obstruction is to pass a flexible suction catheter into the ETT. If obstruction does not resolve after aspiration of secretions, bronchospasm is more likely and should be treated. In some cases, flexible bronchoscopy may be necessary.

Bronchospasm — Bronchospasm during weaning from CPB may be due to an allergic drug reaction (eg, protamine) or transfusion reaction (eg, blood products, plasma expanders), inadequate anesthesia, or preexisting asthma or chronic obstructive pulmonary disease [96]. Bronchospasm is managed by administering bronchodilating pharmacologic agents. Depth of anesthesia is also evaluated because bronchospasm may result from inadequate anesthesia [97]. First-line therapies are inhaled beta$_2$ agonists such as albuterol and/or low doses of intravenous (IV) epinephrine administered initially in 5 to 10 mcg boluses, increasing up to 100 mcg per bolus and/or an infusion at 2 to 10 mcg/minute if necessary (table 6). Also, H1 and H2 antihistamines are administered to reverse the effects of mediator release. In severe cases, steroids such as methylprednisolone or hydrocortisone are administered to decrease airway swelling, stabilize mast cells, and reduce the likelihood of recurrence. (See "Perioperative anaphylaxis: Recognition and emergency management", section on 'Initial management and intravenous epinephrine'.)

If these interventions are not successful, it may be necessary to re-establish CPB.

Pulmonary edema — Cardiogenic pulmonary edema may be due to preexisting or new-onset heart failure exacerbated by additional fluid administration during CPB. Hemoconcentration during CPB and diuresis during and after CPB minimize the likelihood of this complication.

Noncardiogenic pulmonary edema may be caused by protamine, transfusion-related acute lung injury, or other causes of acute respiratory distress syndrome (see "Weaning from cardiopulmonary bypass", section on 'Reversal of anticoagulation with protamine' and "Transfusion-related acute lung injury (TRALI)"). Also, prolonged CPB duration >4 hours is associated with noncardiogenic pulmonary edema due to sequestration of neutrophils in the pulmonary capillaries, elevation of lysosomal enzyme activity, localized inflammatory responses, and, ultimately, increased capillary permeability [98].

Treatment involves the administration of a diuretic such as furosemide and use of lung protective ventilation (see "Mechanical ventilation during anesthesia in adults", section on 'Lung protective ventilation during anesthesia'). In patients with severely elevated pulmonary artery pressure, continuous inhalation of nitric oxide or epoprostenol is reasonable. In rare cases of severe pulmonary edema, extracorporeal membrane

oxygenation (ECMO) is necessary to temporarily replace lung function. (See 'Right ventricular dysfunction' above and "Noncardiogenic pulmonary edema".)

BLEEDING AND COAGULOPATHY

Bleeding necessitating transfusion occurs commonly after cardiac surgery with CPB [99]. Risk factors include preexisting coagulopathy or anticoagulant therapy, advanced age, small body surface area, female gender, need for repeat sternotomy, and urgent or emergency procedures [100]. Specific causes of excessive bleeding during and after weaning from CPB include inadequate surgical hemostasis, loss of platelets and coagulation factors due to persistent surgical bleeding, effects of hemodilution, hypothermic coagulopathy, presence of residual heparin, coagulopathy due to platelet activation (and consumption), and hyperfibrinolysis induced by the extracorporeal circuit. Management of bleeding and coagulopathy after CPB is addressed in detail in a separate topic. (See "Achieving hemostasis after cardiac surgery with cardiopulmonary bypass", section on 'Achieving hemostasis and management of bleeding' and "Point-of-care hemostasis testing (viscoelastic tests)".)

METABOLIC ABNORMALITIES

Metabolic disturbances after CPB are common and are associated with arrythmias and hemodynamic instability.

Hypocalcemia — Hypocalcemia is common following CPB and is corrected with calcium chloride (5 to 10 mg/kg) or, less commonly, calcium gluconate (250 to 1000 mg) during or shortly after separation from CPB. To reduce the risk of reperfusion injury following bypass, treatment is delayed for at least 10 to 15 minutes after removal of the aortic cross-clamp, thus allowing a period of myocardial reperfusion [101].

After separation from CPB, calcium should be immediately available to treat hypocalcemia and/or hyperkalemia. Although calcium chloride transiently improves systolic function and increases systemic vascular resistance (SVR) [102,103], we do not routinely administer calcium after separation from CPB because of the possibility of increased reperfusion injury [104], increased ventricular wall stiffness [103], or induced spasm of the internal mammary artery graft [105].

Hypokalemia — Hypokalemia may contribute to increased cardiac automaticity, leading to the development of atrial or ventricular arrhythmias. Causes of hypokalemia during weaning include preoperative diuretic therapy, mannitol administration during CPB, and treatment of hyperglycemia with insulin administration. Hypokalemia in the postbypass period is corrected by infusion of potassium 10 to 20 mEq over 30 minutes. A central catheter and continuous hemodynamic monitoring are necessary during such rapid administration. (See "Clinical manifestations and treatment of hypokalemia in adults", section on 'Recommended approach'.)

Hyperkalemia — Hyperkalemia may result from potassium cardioplegia or extracellular shifts associated with respiratory or metabolic acidosis. It is more likely to be a clinical problem in patients with impaired kidney function. If severe, it can interfere with cardiac conduction. Potassium level may be decreased during CPB by the performance of “zero-balance” ultrafiltration [106].

A reasonable approach to management of hyperkalemia after CPB includes moderate hyperventilation and, if necessary, administration of calcium, a combination of glucose and insulin, or both. Other treatments include beta-agonists to facilitate intracellular migration of potassium, or furosemide to eliminate potassium via diuresis. (See "Treatment and prevention of hyperkalemia in adults", section on 'Patients with a hyperkalemic emergency'.)

Hypomagnesemia — Hypomagnesemia after CPB may be associated with postoperative dysrhythmias, myocardial ischemia, and ventricular dysfunction [107,108]. Causes include hemodilution with magnesium-free fluids during CPB, as well as diuresis. When hypomagnesemia is confirmed via laboratory analysis or is suspected due to arrhythmias (eg, atrial fibrillation [109]), intravenous (IV) magnesium is administered (1 to 2 g over 15 minutes). Administration of magnesium may result in hypotension [110]. (See "Hypomagnesemia: Evaluation and treatment".)

Hyperglycemia — We agree with the Society of Thoracic Surgeons (STS) guidelines, which recommend maintaining the blood glucose level at <180 mg/dL (10 mmol/L) during CPB and the postbypass period with IV insulin administered as small bolus doses or a continuous insulin infusion if necessary [111]. (See "Glycemic control in critically ill adult and pediatric patients".)

Hyperglycemia is extremely common during and after CPB. Poor perioperative glycemic control has been associated with increased morbidity and mortality [112]. However, attenuating the hyperglycemic response to cardiac surgery and CPB is difficult, and tight control can be harmful [113]. In a randomized trial of 400 cardiac surgical patients receiving either "tight" glucose control during the intraoperative period (ie, use of IV insulin infusion to attempt to maintain intraoperative glucose levels at 80 to 100 mg/dL [4.4 to 5.6 mmol/L]) or conventional management (ie, maintenance of glucose level <200 mg/dL [11.1 mmol/L]), the rate of stroke was significantly higher in the tight-control group [114].

OLIGURIA

Urine output (UO) is measured approximately every 30 minutes in the postbypass period. Ideally, UO is ≥0.5 mL/kg per hour. However, interventions and events occurring during and after CPB may result in increased UO (eg, mannitol) or decreased UO (eg, hemoconcentration, ischemia-reperfusion injury). Also, ongoing effects of anesthesia and surgery may temporarily reduce glomerular filtration, renal tubular function, and UO [115]. If UO is <0.5 mL/kg, we check the bladder catheter for kinking or disconnection, use transesophageal echocardiography (TEE) to exclude aortic dissection as a possible cause of renal hypoperfusion, administer adequate intravascular volume to ensure euvolemia, and maintain adequate cardiac index (CI; ie, >2.0 L/minute per m^2). We do not administer an infusion of "renal dose" dopamine or other pharmacologic agents, as this is not effective for kidney protection [116,117].

HYPOTHERMIA

A decrease in the core temperature after rewarming from hypothermic CPB is termed "afterdrop" and is commonly seen in patients following weaning from CPB [118]. (See "Management of cardiopulmonary bypass", section on 'Temperature'.)

Measures to prevent and treat hypothermia include adequate rewarming prior to separation from CPB (see "Management of cardiopulmonary bypass", section on 'Management during rewarming and weaning'), increasing the room temperature, warming cold blood products during transfusion, and use of patient warming devices that employ forced air or circulating fluid [119]. Supplementary methods such as warming of intravenous (IV) fluids and breathing system humidifiers are less effective [120]. Continuation of active warming is essential for patients who are hypothermic upon arrival in the intensive care unit.

Hypothermia may cause or exacerbate decreased myocardial function due to ischemia, as well as bleeding due to platelet dysfunction, and it decreases metabolism of intravenously administered drugs (see "Perioperative temperature management", section on 'Consequences of hypothermia'). However, temporary mild hypothermia is common postoperatively and appears to have few consequences. Approximately one-half of patients undergoing coronary artery bypass grafting (CABG) surgery with CPB remained hypothermic (<36°C) on arrival to the intensive care unit (ICU) in one observational study [121]. Compared with normothermic patients, a propensity-matched cohort of these hypothermic patients had no differences in postoperative complications (eg, requirement for transfusion of blood products, prolonged duration of controlled ventilation, respiratory complications, gastrointestinal complications, sternal wound infection, sepsis, death).

INABILITY TO CLOSE THE STERNUM

Once hemostasis has been achieved in a patient with hemodynamic and general stability, the sternal retractor is removed, and sternal wires are placed to close the chest. Although sternal closure may cause a slight decrease in the cardiac index (CI) and a slight increase in central venous pressure and/or pulmonary artery pressure owing to compression of the right atrium and ventricle, the surgeon can proceed to close the chest if hemodynamic stability is maintained.

In some patients, particularly those undergoing complex cardiac operations requiring a prolonged duration of CPB, attempted chest closure causes severe hypotension that mandates leaving the sternum open. In these cases, compression of the right atrium and ventricle impairs ventricular filling and decreases CI; these effects are exaggerated in a hypovolemic patient [122]. Attempts to close the sternum may also compromise the surgical repair (eg, kinking of a graft or trapping of a graft in the sternal wall) or cause pacing wire displacement, with resultant hemodynamic instability. Rarely, sternal closure results in entrapment of lung parenchyma or restriction of lung expansion that manifests as increased airway pressure.

If necessary, delayed sternal closure can be performed on a subsequent postoperative day; this is necessary in approximately 4 percent of cardiac surgical cases [123]. Delayed sternal closure eliminates compression of the heart by the sternum, reduces the likelihood of tamponade, and facilitates mediastinal re-exploration if necessary. However, the sternum remains unstable and risk of intrathoracic infection is increased. (See "Surgical management of sternal wound complications".)

The most common indications for delayed sternal closure are low cardiac output syndrome, diffuse bleeding, arrhythmias, tamponade, or cardiac edema. Mortality is higher in patients undergoing delayed sternal closure if ejection fraction is low (<30 percent) with prolonged duration of inotrope requirement, closure is delayed more than six days, or a sternal wound infection develops.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Management of cardiopulmonary bypass" and "Society guideline links: Transfusion and patient blood management".)

SUMMARY AND RECOMMENDATIONS

- **Cardiovascular problems** – Cardiovascular problems that may result in hypotension immediately after cardiopulmonary bypass (CPB) include left ventricular (LV) dysfunction, right ventricular (RV) dysfunction, vasoplegia, arrhythmias, air embolization, LV outflow tract (LVOT) obstruction, and other surgical technical problems. (See 'Cardiovascular problems' above.)
  - We suggest not routinely administering inotropic and vasopressor drugs (**Grade 2B**). However, vasoactive drugs may be necessary to treat low cardiac output syndrome or vasoplegia occurring during CPB and the postbypass period. (See 'Vasoactive drug therapy' above.)
  - In selected patients with postcardiotomy cardiogenic shock, temporary mechanical circulatory support (eg, intraaortic balloon pump [IABP] counterpulsation, percutaneous or implantable ventricular assist device (VAD), extracorporeal membrane oxygenation [ECMO]) may be necessary. Device selection depends on individual patient hemodynamic factors, surgical preferences, and institutional resources. (See 'Cardiogenic shock' above.)
- **Pulmonary problems** – Airway obstruction, bronchospasm, and pulmonary edema may occur in the immediate postbypass period. We suggest a lung protective ventilation strategy in the postbypass period (with low tidal volume [TV] <8 mL/kg ideal body weight, positive end-expiratory pressure [PEEP] ≥5 cmH_2O, and low driving pressure [peak inspiratory pressure – PEEP] <16 cmH_2O) to potentially reduce the incidence of postoperative pulmonary complications (**Grade 2C**). These ventilator settings are consistent with recommendations for lung-protective ventilation for all patients undergoing anesthesia and surgery with use of mechanical ventilation. (See 'Pulmonary problems' above and "Mechanical ventilation during anesthesia in adults", section on 'Lung protective ventilation during anesthesia'.)
- **Bleeding and coagulopathy** – Management of bleeding and coagulopathy after CPB is addressed separately. (See "Achieving hemostasis after cardiac surgery with cardiopulmonary bypass", section on 'Achieving hemostasis and management of bleeding'.)
- **Metabolic disturbances**
  - **Electrolyte abnormalities** – Hypocalcemia, hypokalemia, hyperkalemia, or hypomagnesemia are common and may be associated with arrythmias and hemodynamic instability. (See 'Metabolic abnormalities' above.)
  - **Hyperglycemia** – We suggest maintaining the blood glucose level at <180 mg/dL (10 mmol/L) during CPB and the postbypass period (**Grade 2C**). Treatment is with intermittent doses of insulin if effective, or a continuous insulin drip if glucose levels are persistently elevated. We also recommend avoiding a more stringent target (eg, 80 to 100 mg/dL [4.4 to 5.6 mmol/L]) (**Grade 1A**). (See 'Hyperglycemia' above.)

- **Hypothermia** – Hypothermia is associated with arrhythmias, increased bleeding, and delayed extubation. We employ measures to prevent and treat postbypass hypothermia including adequate rewarming prior to separation from CPB, increasing the room temperature, warming cold blood products and intravenous (IV) fluids, and use of patient warming devices that employ forced air or circulating fluid. (See 'Hypothermia' above.)

- **Inability to close the sternum** – Occasionally, attempted chest closure causes severe hypotension. Causes include compression of the right atrium and RV that impairs ventricular filling or compromise of the surgical repair (eg, kinking of a graft or trapping of a graft in the sternal wall). In such patients, delayed sternal closure can be performed on a subsequent postoperative day. (See 'Inability to close the sternum' above.)

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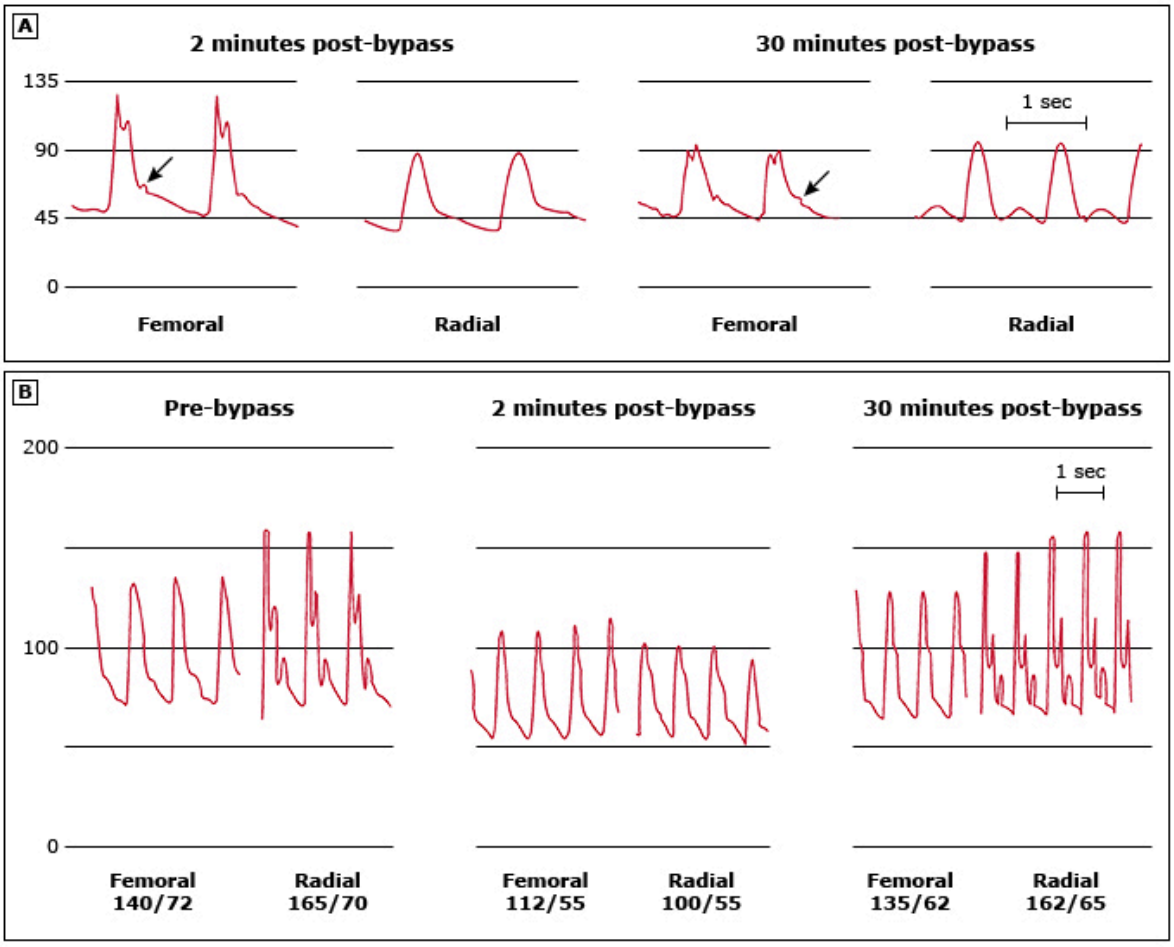
Topic 115472 Version 38.0

GRAPHICS

Figure 1: **Central to peripheral arterial pressure gradients after cardiopulmonary bypass**



Arterial pressure gradients following cardiopulmonary bypass.
(A) Femoral and radial arterial pressure tracings recorded two minutes after bypass (2 minutes post-bypass), when radial arterial pressure underestimates the more centrally measured femoral arterial pressure and 30 minutes later (30 minutes post-bypass), when radial and femoral arterial pressures have equalized and radial pressure has resumed a more typical morphology. Note that dicrotic notch (arrows) is visible in the femoral pressure tracing immediately after bypass, but is delayed in the radial pressure tracing.
(B) Femoral and radial arterial pressure tracings recorded before cardiopulmonary bypass (pre-bypass), two minutes following bypass (2 minutes post-bypass), and 30 minutes following bypass (30 minutes post-bypass). Note changing relationship between femoral and radial arterial pressure measurements at these different times.

Reproduced from: Schroeder R, Barbeito A, Bar-Yosef S, Mark JB. Cardiovascular monitoring. In: Miller's Anesthesia, 8th ed, Miller RD, Eriksson LI, Fleisher LA, et al (Eds), Saunders 2014. p.1345. Illustration used with the permission of Elsevier Inc. All rights reserved.

Graphic 111035 Version 1.0

Table 1: **Hemodynamic management of hypotension during and after weaning from CPB**

CVP	PAP	PADP or PAWP*	Cardiac output	Blood pressure	LV function by TEE	RV function by TEE	Diagnosis	Response
Low	Low	Low	Low	Low	Normal or hyperdynamic	Normal or hyperdynamic	Hypovolemia	Administer volume
Low or normal	Low or normal	Low or normal	High	Low	Normal or hyperdynamic	Normal or hyperdynamic	Vasoplegia	Administer vasopressor
High or normal	High	Low or normal¶	High	Low	Normal	Normal or hypocontractile	Pulmonary hypertension	Administer pulmonary vasodilator
High	Normal or low	Normal or high¶	Low	Low	Normal	Hypocontractile, often dilated	Right ventricular dysfunction	Administer inotropic agent Administer pulmonary vasodilator
Normal or high	Normal or high	High	Low	Low	Hypocontractile, often dilated	Normal	Left ventricular dysfunction	Administer inotropic agent

CPB: cardiopulmonary bypass; CVP: central venous pressure; PAP: pulmonary artery pressure; PADP: pulmonary artery diastolic pressure; PAWP: pulmonary artery wedge pressure; LV: left ventricular; TEE: transesophageal echocardiography; RV: right ventricular.
* PAWP should not be measured prior to neutralizing heparin following CPB. Initially, PADP is measured, the PADP may overestimate PAWP when patients have elevated pulmonary vascular resistance (eg, pulmonary hypertension).
¶ PADP or PAWP are indirect measures of LV filling pressure. With RV dysfunction and dilation, ventricular septal shift may increase LV filling pressure despite low or normal LV filling volume.

Graphic 93595 Version 8.0

Table 2: **Vasopressors and inotropic agents used in the operating room: Adult dosing** *¶

Drug	Functional class (predominant receptor or mechanism of action)	Bolus dose	Infusion dose	Comments
Ephedrine	Inotrope/chronotrope/vasopressor (alpha$_1$-adrenergic receptor agonist; beta$_1$- and beta$_2$-adrenergic receptor agonist)	5 to 10 mg boluses	N/A	▪ Tachyphylaxis may occur with multiple repeated doses due to indirect postsynaptic release of norepinephrine ▪ Cardiovascular effects attenuated by drugs that block ephedrine uptake into adrenergic nerves (eg, cocaine) or those that deplete norepinephrine reserves (eg, reserpine) ▪ Administered with extreme caution (eg, in small incremental doses of 2.5 mg) to patients using monoamine oxidase (MAO) inhibitors or methamphetamines since exaggerated hypertensive responses or life-threatening dysrhythmias may occur
Phenylephrine	Vasopressor (alpha$_1$-adrenergic receptor agonist)	50 to 100 mcg boluses (may begin infusion if repeated bolus doses are necessary)	10 to 100 mcg/minute **or** 0.1 to 1 mcg/kg/minute	▪ Often selected to treat hypotension if normal or elevated HR is present ▪ Genetic polymorphisms lead to variable individual responses
Norepinephrine	Inotrope/vasopressor (alpha$_1$- and beta$_1$-adrenergic receptor agonist)	4 to 8 mcg (may begin infusion if repeated bolus doses are necessary)	1 to 20 mcg/minute **or** 0.01 to 0.3 mcg/kg/minute	▪ Often selected as a first-line agent during noncardiac surgery, particularly for treatment of most types of shock

				▪ Norepinephrine 8 mcg is approximately equivalent in potency to phenylephrine 100 mcg ▪ Peripheral extravasation of a high concentration may cause tissue damage
Epinephrine	Inotrope/chronotrope/vasopressor (alpha$_1$-adrenergic receptor agonist; beta$_1$- and beta$_2$-adrenergic receptor agonist)	4 to 10 mcg initially; up to 100 mcg boluses may be used when initial response is inadequate	1 to 100 mcg/minute **or** 0.01 to 1 mcg/kg/minute **Note changing effects across dose range:** ▪ Low doses have primarily beta$_2$-adrenergic effects at 1 to 2 mcg/minute or 0.01 to 0.02 mcg/kg/minute ▪ Intermediate doses have primarily beta$_1$- and beta$_2$-adrenergic effects at 2 to 10 mcg/minute or 0.02 to 0.1 mcg/kg/minute ▪ High doses have primarily alpha$_1$-adrenergic effects at 10 to 100 mcg/minute or 0.1 to 1 mcg/kg/minute	▪ First-line treatment for cardiac arrest and for anaphylaxis ▪ May be administered IV, IM, or via an endotracheal tube in emergencies ▪ Low doses cause bronchodilatory effects and may cause arterial vasodilation and decreased BP ▪ Intermediate doses cause increases in HR and BP ▪ High doses cause vasoconstriction, with possible severe hypertension and adverse metabolic effects ▪ Individual responses to dose-related effect are variable
Vasopressin	Vasopressor (vasopressin$_1$ and vasopressin$_2$ receptor agonist)	1 to 4 units	0.01 to 0.04 units/minute Doses >0.04 units/minute up to 0.1 units/minute are reserved for salvage therapy (ie, failure to achieve adequate BP goals with other vasopressor agents)¶	▪ Effective for treatment of hypotension refractory to administration of catecholamines or sympathomimetics such as ephedrine, phenylephrine, or norepinephrine ▪ No direct effect on HR ▪ Little effect on PVR; can cause splanchnic

				vasoconstriction ▪ Individual responses to dose-related effects are variable ▪ Peripheral extravasation may cause skin necrosis
Dopamine	Inotrope/vasopressor/dose-dependent chronotropy (dopaminergic, $beta_1$-, $beta_2$-, and $alpha_1$-adrenergic receptor agonist)	N/A	2 to 20 mcg/kg/minute **Note changing effects across dose range:** ▪ Low doses have primarily dopaminergic effects at <3 mcg/kg/minute ▪ Intermediate doses have primarily $beta_1$- and $beta_2$-adrenergic effects at 3 to 10 mcg/kg/minute ▪ High doses have primarily $alpha_1$-adrenergic effects >10 mcg/kg/minute	▪ Low doses may exacerbate hypotension via $beta_2$ stimulation ▪ High doses may cause vasoconstriction, adverse metabolic effects, and arrhythmias
Dobutamine	Inotrope/vasodilator/dose-dependent chronotropy ($beta_1$- and $beta_2$-adrenergic receptor agonist)	N/A	1 to 20 mcg/kg/minute	▪ Exacerbation of hypotension is possible due to dose-dependent vasodilation (via $beta_2$ stimulation); concurrent administration of a potent vasoconstrictor such as norepinephrine or vasopressin may be necessary
Milrinone	Inotrope/vasodilator (phosphodiesterase inhibitor) (decreases rate of cyclic adenosine monophosphate [cAMP] degradation)	N/A	0.375 to 0.75 mcg/kg/minute (a loading dose of 50 mcg/kg over ≥10 minutes may be administered, but may be omitted to avoid hypotension)	▪ Exacerbation of hypotension is likely due to vasodilation (via phosphodiesterase inhibition); concurrent administration of a potent vasoconstrictor such as norepinephrine or

				vasopressin may be necessary
Isoproterenol	Inotrope/chronotrope/vasodilator (beta$_1$- and beta$_2$-adrenergic receptor agonist)	N/A	5 to 20 mcg/minute **or** 0.05 to 0.2 mcg/kg/minute	▪ Exacerbation of hypotension is likely due to dose-dependent vasodilation (via beta$_2$ stimulation) ▪ May cause arrhythmias ▪ Not available in most settings
Levosimendan	Inotrope/vasodilator(calcium sensitizer; phosphodiesterase inhibition)	N/A	Initial dose: 6 to 12 mcg/kg infused over 10 minutes. Maintenance dose: 0.05 to 0.2 mcg/kg/minute by continuous infusion	▪ Short-term effects include dose-dependent reduction in cardiac filling pressures, increase in cardiac index, and vasodilation ▪ May have favorable effects on relaxation properties in failing hearts ▪ Not available in the United States

BP: blood pressure; HR: heart rate; IM: intramuscular; IV: intravenous; N/A: not applicable; PVR: pulmonary vascular resistance.

* Dose ranges are based on adult patients of average size.

¶ Refer to related UpToDate content on hemodynamic management during anesthesia and surgery.

Graphic 119747 Version 5.0

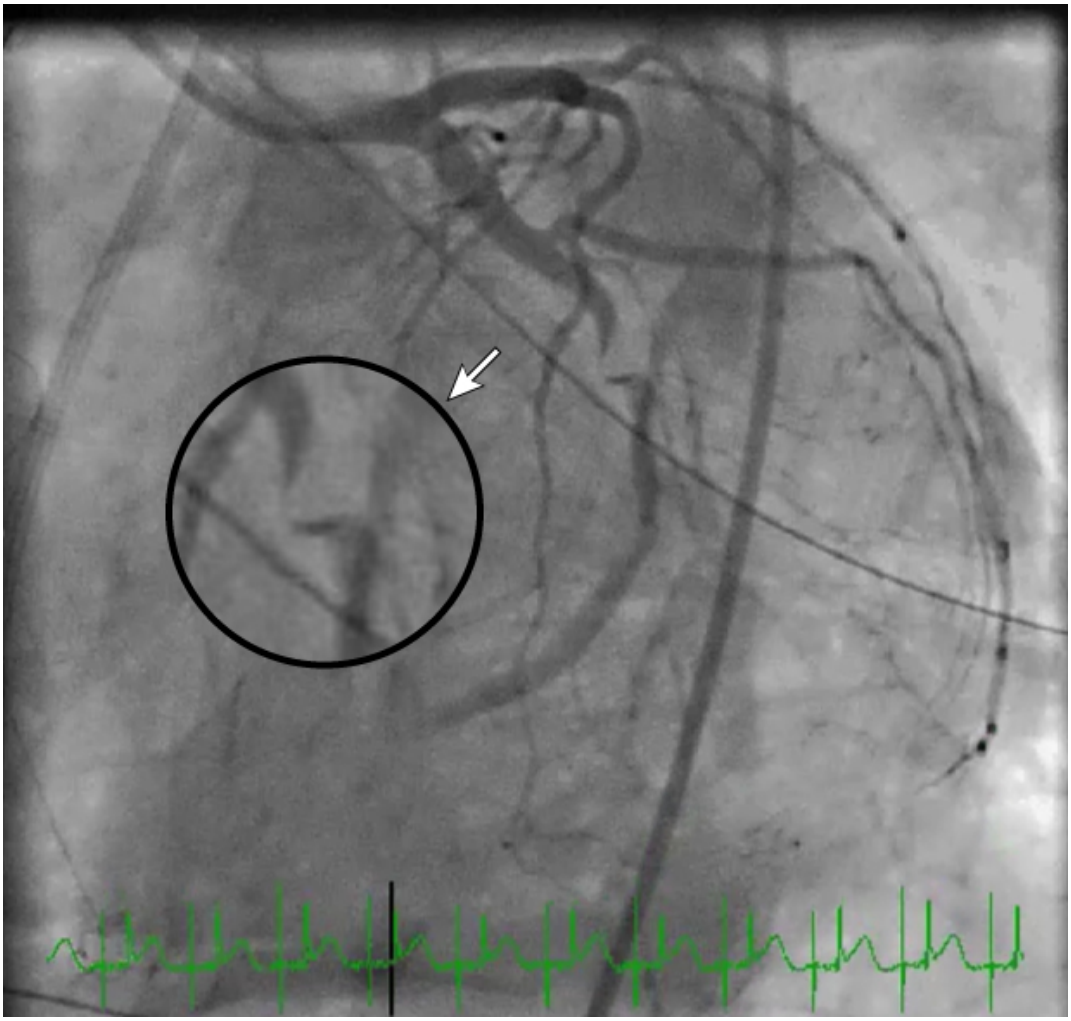
Table 3: **Vasopressors used to treat vasoplegic syndrome**

Drug	Dose	Description
Norepinephrine	1 to 20 mcg/minute or 0.01 to 0.3 mcg/kg/minute	$Alpha_1$- and $beta_1$-adrenergic receptor agonist; adrenergic-mediated vascular smooth muscle vasoconstriction and beta-receptor-mediated release of AT-II in kidneys
Vasopressin	0.01 to 0.04 units/minute	$Vasopressin_1$ and $vasopressin_2$ receptor agonist; direct vascular smooth muscle vasoconstriction and increased permeability of collecting ducts to water
Phenylephrine	10 to 100 mcg/minute or 0.1 to 1 mcg/kg/minute	$Alpha_1$-selective adrenergic receptor agonist; adrenergic-mediated vascular smooth muscle vasoconstriction
Methylene blue	1 to 2 mg/kg IV over 10 to 20 minutes, followed by 0.5 to 1 mg/kg/hour infusion	Inhibits NOS and GC activation to inhibit vascular smooth muscle relaxation
Angiotensin II	Start at 10 to 20 ng/kg/minute, then titrate every 5 minutes by up to 15 ng/kg/minute increments to a maximum dose of 80 ng/kg/minute for the first 3 hours. The maximum maintenance dose should not exceed 40 ng/kg/minute.	Octapeptide that causes venous and arterial smooth muscle vasoconstriction and acts on the CNS to increase vasopressin production
Hydroxocobalamin	5 g IV over 5 to 10 minutes	Vitamin B_{12} form that causes direct binding of NO and inhibitory effects on NOS and GC to inhibit vascular smooth muscle relaxation

AT-II: angiotensin II; IV: intravenous; NOS: nitric oxide synthase; GC: guanylyl cyclase; CNS: central nervous system; NO: nitric oxide.

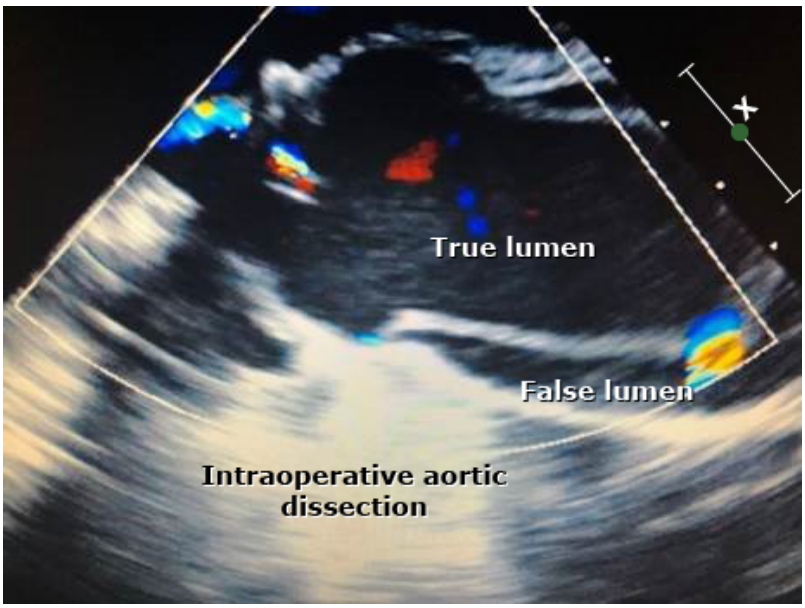
Graphic 126194 Version 3.0

Image 1: **Angiogram showing inadvertent ligation of the left circumflex coronary artery noted after cardiac surgery with cardiopulmonary bypass**



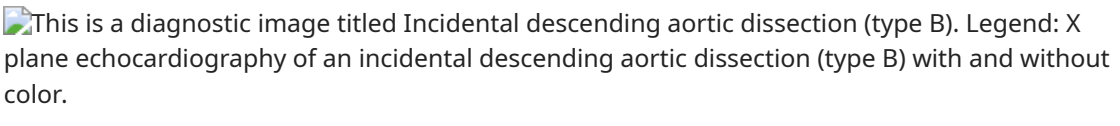
Graphic 148525 Version 1.0

Image 2: **Transesophageal echocardiograph image showing iatrogenic aortic dissection**



Graphic 120106 Version 1.0

Image 3: **Incidental descending aortic dissection (type B)**


This is a diagnostic image titled Incidental descending aortic dissection (type B). Legend: X plane echocardiography of an incidental descending aortic dissection (type B) with and without color.

X plane echocardiography of an incidental descending aortic dissection (type B) with and without color.

Graphic 134844 Version 1.0

Image 4: **TEE (ME LAX view) showing hypertrophic cardiomyopathy (HCM) with systolic anterior motion (SAM) of the anterior leaflet of the mitral valve**

This is a diagnostic image titled TEE (ME LAX view) showing hypertrophic cardiomyopathy (HCM) with systolic anterior motion (SAM) of the anterior leaflet of the mitral valve. Legend: TEE midesophageal long-axis view of a patient with hypertrophic cardiomyopathy and SAM with LVOT obstruction. The arrow shows the point of mitral-septal contact.

TEE midesophageal long-axis view of a patient with hypertrophic cardiomyopathy and SAM with LVOT obstruction. The arrow shows the point of mitral-septal contact.

TEE: transesophageal echocardiography; ME: midesophageal; LAX: long-axis; HCM: hypertrophic cardiomyopathy; SAM: systolic anterior motion of the mitral valve; LVOT: left ventricular outflow tract.

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Graphic 102345 Version 5.0

Table 4: **Temporary mechanical devices for cardiac support**

Device	Maximum support (L/minute)	RV support	Support duration	Percutaneous implantation	Speed to establish	Contraindications
IABP[1]	0.5	No	Weeks	Yes	Quick	▪ AI ▪ Aortic dissection ▪ Abdominal aortic aneurysm
Tandem Heart[2]	5	No	Four weeks	Yes	Moderate	▪ Moderate to severe AI ▪ Ventricular septal defect
Centrimag (VAD)[3]	10	Yes	Months	Yes	Slow	
ECMO	6	Yes	Weeks	Yes	Quick	▪ Prolonged mechanical ventilation (>7 days) ▪ Moderate to severe AI
Impella CP[4]	>4.3	No	<4 days	Yes	Quick	▪ LV thrombus ▪ Mechanical AoV ▪ Severe AS ▪ VSD ▪ Significant right heart failure ▪ Severe PAD ▪ Combined cardiorespiratory failure
Impella 5.5[5]	5.5	No	Weeks	Yes	Quick	
Impella RP[6]	>4	Yes	Weeks	Yes	Quick	▪ Pulmonary artery abnormalities (thrombus, aneurysm) ▪ Significant TV or PV stenosis ▪ TV and PV prosthetic mechanical valves ▪ Significant PV regurgitation ▪ Right sided thrombus ▪ IVC filter (Impella RP) ▪ SVC abnormalities (Impella RP Flex)
Impella RP Flex	>4	Yes	Weeks	Yes	Quick	

AI: aortic insufficiency; AoV: aortic valve; AS: aortic stenosis; ECMO: extracorporeal membrane oxygenation; IABP: intra-aortic balloon pump; IVC: inferior vena cava; LV: left ventricular; PAD: peripheral arterial disease; PFO: patent foramen ovale; PV: pulmonic valve; RV: right ventricular; TV: tricuspid valve; VAD: ventricular assist device; VSD: ventricular septal defect.

References:

1. *Kantrowitz A, Tjonneland S, Freed PS, et al. Initial clinical experience with intraaortic balloon pumping in cardiogenic shock. JAMA 1968; 203:113.*
2. *TandemHeart. https://www.livanova.com/advanced-circulatory-support/en-us/tandemheart-kit (Accessed on May 14, 2024).*
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4. *Impella CP. https://www.abiomed.com/products-and-services/impella/impella-cp-with-smartassist (Accessed on November 16, 2023).*
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6. *Impella RP. https://www.abiomed.com/products-and-services/impella/impella-rp-with-smartassist (Accessed on November 16, 2023).*

Graphic 116235 Version 5.0

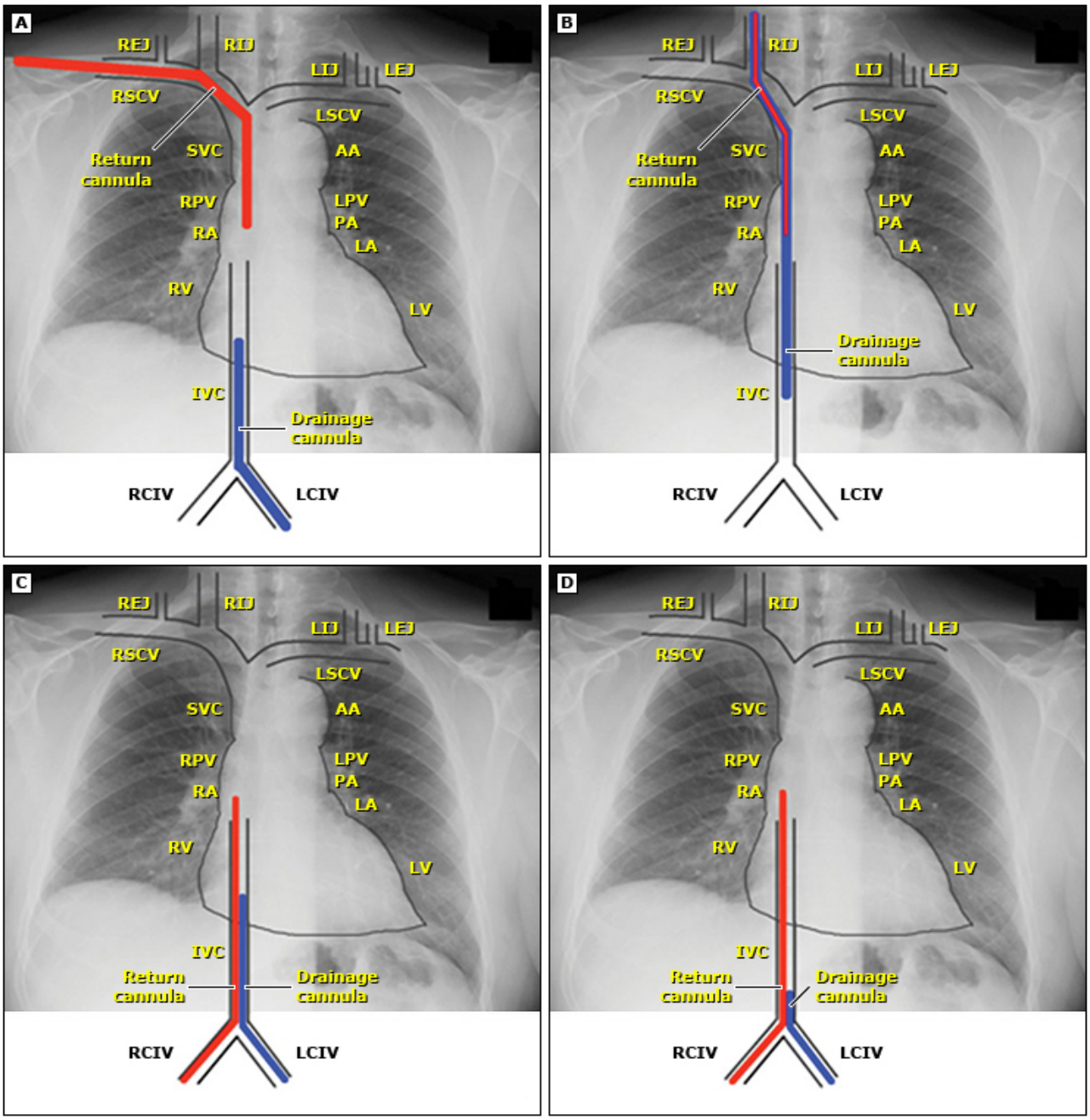
Table 5: **Contraindications and complications for all temporary mechanical support devices**

Contraindications for ALL temporary mechanical support devices	Complications associated with ALL temporary mechanical support devices
Severe peripheral vascular disease	Bleeding
Irreversible neurological disease	Vascular injury
Sepsis (relative)	Infection
Patient or proxy refusal	Neurologic injury
Contraindication to anticoagulation	Displacement and malposition

Adapted from: Gilotra NA, Stevens GR. Temporary Mechanical Circulatory Support: A Review of the Options, Indications, and Outcomes. Clin Med Insights Cardiol 2014; 8(Suppl 1):75.

Graphic 119277 Version 1.0

Image 5: **Common venovenous ECMO cannulation strategies**



Venovenous ECMO relies upon drainage of venous blood from the body and then a return of blood that has been oxygenated to the venous circulation. If the sites of venous drainage and return of oxygenated blood are too close, mixing can occur between the cannulae. Thus, the tips of the two cannula should be separated.

Blue lines denote drainage cannulas, which deliver venous blood from the patient to the membrane oxygenator. Red lines denote return cannulas, which deliver oxygenated blood from the membrane oxygenator to the patient.

(A) Femoral-right internal jugular cannulation.

(B) Dual-lumen right internal jugular cannulation with Avalon Elite cannula. With this cannula, drainage occurs in the SVC and IVC, and blood is returned to the RA.

(C) Femoral-femoral cannulation with the return cannula at the junction of the IVC and RA and drainage in the IVC.

(D) Femoral-femoral cannulation with the return cannula in the RA and the drainage cannula in the femoral vein and distal IVC. Femoral cannulae are inserted into the femoral vein, which drains into the common iliac vein.

ECMO: extracorporeal membrane oxygenation; REJ: right external jugular vein; RIJ: right internal jugular vein; LIJ: left internal jugular vein; LEJ: left external jugular vein; RSCV: right subclavian vein; LSCV: left subclavian vein; SVC: superior vena cava; AA: aortic arch; RPV: right pulmonary vein; LPV: left pulmonary vein; RA: right atrium; PA: pulmonary artery; LA: left atrium; RV: right ventricle; LV: left ventricle; IVC: inferior vena cava; RCIV: right common iliac vein; LCIV: left common iliac vein.

From: Fierro MA, Daneshmand MA, Bartz RR. Perioperative management of the adult patient on the venovenous extracorporeal membrane oxygenation requiring noncardiac surgery. Anesthesiology 2018; 128:181. DOI: 10.1097/ALN.0000000000001887. Copyright © 2018 American Society of Anesthesiologists. Reproduced with permission from Wolters Kluwer Health. Unauthorized reproduction of this material is prohibited.

Graphic 116518 Version 5.0

Table 6: **Emergency management of anaphylaxis in the perioperative setting (adult)**

Signs (and symptoms, if patient can communicate)
▪ Hypotension/shock ▪ Tachycardia/arrhythmias ▪ Dyspnea/wheezing/high peak inspiratory pressure/bronchospasm ▪ Hypoxemia or decreased O_2 saturation noted on pulse oximetry ▪ Angioedema (face, lips, laryngeal stridor ▪ Hives/itching ▪ Flushing
Immediate actions
▪ Call for help. Inform team and surgeon. Get code cart. ▪ Check for possible causes – Stop antibiotics, muscle relaxants, IV contrast, blood products, and latex exposure. ▪ Administer 100% FiO_2. ▪ Secure or establish airway. ▪ For hypotension/shock, give epinephrine bolus (may repeat or escalate dose in 1 to 2 minutes and titrate to effect). • Dosing 10 to 100 mcg per IV bolus. • If no detectable blood pressure, then initiate ACLS protocol and administer fluid bolus (approximately 25 to 50 mL/kg). ▪ For ongoing hypotension, start IV infusion of epinephrine. • Dosing 2 to 10 mcg/minute, titrated to effect.
Other therapies for anaphylaxis (if signs/symptoms persist)
▪ Albuterol 4 to 8 puffs initially for bronchospasm; alternatively, 2.5 mg albuterol in 2.5 mL of saline nebulized for administration via the endotracheal tube. ▪ Methylprednisolone 125 mg IV or hydrocortisone 100 mg IV. ▪ H1 antihistamines – Diphenhydramine 50 mg IV. ▪ H2 antihistamines – Famotidine 20 mg IV. ▪ For hypotension that remains refractory during epinephrine administration, add another vasopressor: • Vasopressin (1 to 2 units bolus and/or infusion) or • Norepinephrine infusion (2 to 10 mcg/minute titrated to effect) ▪ Continue fluid administrations for refractory hypotension up to 50 mL/kg. After the first 2 liters or fluid resuscitation for refractory shock, further hemodynamic monitoring should be considered (eg, echocardiography [transthoracic or transesophageal]) to diagnose cause(s). ▪ If patient has received high-dose beta blockers, consider vasopressin infusion or glucagon (1 to 5 mg slow IV bolus over 5 minutes) for refractory shock.
Consider and exclude other causes of signs/symptoms
▪ Pneumothorax/tension pneumothorax ▪ Pericardial tamponade ▪ Myocardial infarction ▪ Aspiration ▪ Pulmonary edema ▪ Air or pulmonary embolus

If patient develops cardiovascular collapse

- Go to ACLS protocols for:
  - Asystole/PEA
  - VF/VT
  - No detectable blood pressure

Send serum tryptase

- Obtain at least 1 mL of blood in a red top tube for serum tryptase assay, ideally within 1 hour, but may remain elevated up to 3 hours from onset of event. Repeat serum tryptase after 24 hours to establish baseline concentration.

After resuscitation

- Observe patient in the intensive care unit or recovery room for 12 to 24 hours.
- Continue glucocorticoids for 24 hours.
- Refer to allergist.

ACLS: advanced cardiac life support; FiO_2: fraction of inspired oxygen; IV: intravenous; O_2: oxygen; PEA: pulseless electrical activity; VF: ventricular fibrillation; VT: ventricular tachycardia.

Graphic 107933 Version 12.0

Contributor Disclosures

Michael G Fitzsimons, MD No relevant financial relationship(s) with ineligible companies to disclose. **Hilary P Grocott, MD, FRCPC** Equity Ownership/Stock Options: Anesthesia Labs, Inc. [IV infusion pumps]. All of the relevant financial relationships listed have been mitigated. **Nancy A Nussmeier, MD, FAHA** No relevant financial relationship(s) with ineligible companies to disclose.

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